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Clinical presentation, diagnosis, complications and treatment of obstructive sleep apnea syndrome

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ABSTRACT

Obstructive sleep apnea syndrome (OSAS) is a disorder characterized by recurrent obstruction of the upper airway during sleep. The most common nocturnal symptom is snoring, and the most common daytime symptom is excessive sleepiness. OSAS causes serious morbidity and mortality due to its cardiovascular, social and neuropsychological consequences. Early diagnosis and treatment are needed at all ages.

Keywords: Sleep, obstructive sleep apnea, diagnosis, treatment

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a syndrome characterized by recurrent complete or partial upper airway obstruction during sleep and frequently decreased blood oxygen saturation.[1] It is a respiratory-related sleep disorder with a high prevalence and often overlooked. It is characterized by frequent awakenings during the night, hypoxemia, sleep disruption and decreased sleep quality.[2] Apneas are classified into 3 groups according to the presence of respiratory effort: obstructive, central and mixed. Obstructive apnea is the presence of respiratory effort despite the absence of airflow in the mouth and nose; central apnea is the absence of both airflow and respiratory effort; and mixed apnea is the continuation of apnea, which is initially of the central type, despite the onset of respiratory effort.[2,3] In adults, apnea is the complete cessation of airflow in the mouth and nose for at least 10 seconds. Hypopnea is a decrease in airflow of at least 30% for 10 seconds or longer and a 3% decrease in oxygen saturation or arousal.[2-4]

PHYSIOPATHOLOGY IN OSAS

Since 2023, the new scoring rules revised by the American Academy of Sleep Medicine (AASM) have been applied.[5] The most important feature of sleep apnea is the repetitive inhibition of respiration by collapse of the upper airway (URTI) during sleep.[6,7] OSAS is a pathologic condition in which recurrent narrowing or closure of the upper airway during sleep causes complete cessation of airflow (apnea) and/or partial (>50%) reduction of airflow (hypopnea) for at least 10 seconds, leading to a minimum 4% decrease in blood oxygen saturation level (SAO_2).[2,6-8] OSAS was described by Guilleminault et al.[9] in 1976.

In the physiopathology of OSAS, narrowing of the lateral diameter of the upper airway and decrease in muscle activity play an important role. The development of upper airway obstruction depends on three main factors. These are the tone of the pharyngeal muscles, the negative pressure during inspiration and the anatomy of the upper airway.[7] The patency of the upper airway is maintained by the activity of the upper airway dilator muscles against the collapsing effect of the negative intraluminal pressure during inspiration. The pressure reaching the upper airways during breathing is counteracted by the muscles and no collapse of the upper airway occurs. If there is a pathology in the activity of these muscles, apnea develops as a result of collapse.[10,11] Pathologies that narrow the upper airway extending from the tip of the nose and mouth to the trachea (large uvula, tonsillar hypertrophy, retrognathia, hypertrophy of the tongue root, plicae forming stenosis in the pharyngeal mucosa), anteroposteror disharmony cause more negative pressure for inspiration. Thus, obstructive apnea occurs.[11] After deep sleep and a further decrease in muscle tone, the accelerated inspiratory air in the narrowed upper airway creates more negative pressure and suction force on the airway wall. When the suction force exceeds the muscle tone trying to keep the airway open, the airway in that area collapses and apnea develops. In patients with OSAS, negative pressure is countered by the activities of the m.genioglossus and m.tensor

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palatini muscles during wakefulness. In sleep, compensation is lost, and symptoms appear.[11]

In addition, orexin, also known as hypocretin, is a neurotransmitter that maintains alertness and stimulates feeding. Orexin by glucose and leptin. Leptin is an anorexigenic hormone that gives a feeling of fullness in normal people. It has been shown that patients with OSAS have leptin resistance. The inhibitory effect of leptin on orexin and the function of orexin in maintaining wakefulness are thought to play a role in the development of sleep in OSAS.[12]

PHYSICAL EXAMINATION IN PATIENTS WITH OSAS

Detailed physical examination is of great importance in the selection of patients for PSG to identify individuals at risk for OSAS. In determining the severity of OSAS, neck circumference measurement and body-mass index (BMI) calculation, which are highly correlated with apnea hypopnea index (AHI), should be included in routine physical examination.[13] A neck circumference above 43 cm in men and above 40 cm in women constitutes a significant risk for OSAS. In most studies, waist/hip ratio has been shown to be a better predictor in individuals diagnosed with OSAS. Maxiofacial and cephalometric measurements can also be performed.[8]

RADIOLOGY IN PATIENTS WITH OSAS

Radiology is not routinely required in patients with OSAS. In these patients, radiology is used to evaluate and decide on some treatment approaches such as intraoral appliances or skeletal surgery.[9]

RISK FACTORS IN PATIENTS WITH OSAS

Gender

Although male gender is considered to be an important risk factor, recent studies have reported that OSAS can be seen at a high rate especially in premenopausal and obese women and the female/male ratio is approximately 1/3 for each age group. Pharyngeal anatomical structures, fat distribution, craniofacial dimensions and male hormones of men are more prone to obstruction formation.[2,7,14]

Age

It is usually seen in children between 2-6 years of age. It is less common in children who have undergone tonsillectomy. The risk increases between the ages of 10-60 and with increasing age. There is a two-fold increase in incidence every ten years. After the age of 60, the incidence continues to increase but the severity of the clinic decreases.[4,7]

Obesity there is a definite relationship between obesity and apnea formation. Obesity increases the risk of OSAS 10-fold in the middle-aged population.[15] The most commonly used parameter to assess the degree of obesity is body-mass index (BMI). In studies, it is known that high BMI leads to some sleep problems.[2,7,14,16]

Anatomical Risk Factors

Craniofacial anomalies such as retrognathia or micrognathia and nasal septum deviation narrow the upper airway passage and cause sleep apnea. Airway length, neck diameter (>43 cm in men, >38 cm in women, head-neck position, dynamic upper respiratory tract (URT) collapse. Abnormal soft tissue mass in the upper airway due to fat deposition or large tonsils is another cause of obstruction. In addition, sleeping in the supine position may increase the severity of sleep apnea by causing the tongue root to obstruct the URT.[17]

Genetics

OSAS is more common in relatives of patients than in normal people. There are studies showing that it is associated with many congenital diseases (Fragile x, Trisomy 21, Marfan syndrome). Sleep disorders are associated with many genes and show complex inheritance characteristics in which environmental interaction is more frequent. Genetic association is common in obstructive sleep apnea syndrome, narcolepsy and restless legs syndrome.[18]

Alcohol

Decreases pharyngeal dilator muscle activity by suppressing neurological stimulation. Thus, pharyngeal collapse is facilitated. In addition, alcohol increases pharyngeal and nasal resistance with its irritant and vasodilator effect on mucous membranes. This leads to an increase in pleural and pharyngeal negative pressure, again facilitating upper airway collapse. Individuals with apnea should stop drinking alcohol 4-6 hours before going to sleep. It has been reported that the number and frequency of apneas are more severe in the first hour of sleep after alcohol intake.[4,7] While Wisconsin found an independent association between OSAS and smoking, Stardling and Crosby found no association between smoking and OSAS.[4]

Individuals with OSAS should also avoid the use of sedative and hypnotic drugs. Sedatives selectively decrease the activity of the nervus hypoglossus and nervus recurrens. These nerves innervate the genioglossus and posteror cricoarytenoid muscles, muscles that play an important role in maintaining rigidity of the oropharynx and larynx. This facilitates upper airway collapse.[11]

Race

It is more common in Asians and African-Americans.[4]

Neuromuscular and Mechanical Factors

URS dilator muscles, dilator muscle-diaphragm relationship, upper airway reflexes lead to OSAS. Airway diameter and shape, lying position, URI resistance, URI compliance, intraluminal pressure, extraluminal pressure, mucosal adhesive effects and vascular factors play a role.[9]

Central Factors

Hypocapneic threshold, periodic breathing, arousal, cytokines are important.[19]

CLINICAL DIAGNOSIS

The three major symptomatic manifestations of the disease are snoring, diagnosed apnea and excessive daytime sleepiness (Table 1).[3]

Snoring

It is the sound caused by noisy vibration in the oropharynx upper airways during inspiration during sleep. It is one of the most common symptoms in the diagnosis of OSAS and is present in 70-95% of cases.[20] Snoring is prominent in two

Table 1. Daytime and nocturnal findings associated with OSAS

Findings associated with OSAS*	
Day	**Night**
• Witnessed apnea • Snoring • Waking up without rest • Night sweats • Don't be thirsty at night • Nokturi • Insomnia**	• Excessive daytime sleepiness • Traffic, home, work accident • Fatigue • Morning headache • Impaired concentration • Irritability • Mood disorders • Decreased libido • Impotence

*OSAS: Obstructive sleep apnea syndrome, **Inability to stay asleep or wake up and go back to sleep

disease groups such as upper airway resistance syndrome (UARS) and OSAS. Most patients with OSAS snore at least 5 nights a week and sleep are interrupted by frequent recurrent apneas.[21]

Witnessed Apnea

It can often be recognized by a relative or intimate partner.[12] Excessive Daytime Sleepiness (EDS).[15] The American Academy of Sleep Medicine (AASM) defines ODSS as the inability to maintain wakefulness during certain waking periods of the day, with sleep occurring involuntarily or at inappropriate times for at least 3 months.[5] The common daytime symptoms of OSAS are GASD (feeling very sleepy at times or completely) and fatigue (feeling low energy and unmotivated).[15] The pathogenesis of EDS is still controversial. It has been reported that nocturnal hypoxemia and autonomic arousal may play an important role in the development of GAUH. There is evidence that chronic intermittent hypoxia and oxidative stress lead to neuronal brain damage resulting in EDS.[12] Apnea duration, decreases in nocturnal 02 saturation, cardiac autonomic dysregulation, and increased sleep fragmentation level are factors that affect EDS.[12] The prevalence of EDS is around 18%, with 13% in men and 6% in women. EDS has been found to be higher especially in patients with severe OSAS compared to the normal population.[22,23] It has been reported that the AHI index is the strongest predictor of the ELS score.[4,22] However, EDS in OSAS is not always correlated with AHI.[12] EDS is a symptom with low specificity because it can be seen in many acute and chronic diseases other than OSAS. OSAS-related EDS can be improved in more than 90% of patients receiving continuous positive airway pressure (CPAP) therapy.[22]

Several scales and questionnaires have been developed to help select patients for polysomnography (PSG) to predict the diagnosis of OSAS. These include the Epworth Sleepiness Scale (ESS), a subjective test in OSAS, or the Maintenance of Wakefulness test (MWT) and Multiple Sleep Latency Test (MSLT), objective tests. The EDS is the most commonly used scale for the determination of excessive daytime sleepiness.[6] Although the EDS is the most widely used questionnaire, the STOP-BANG questionnaire is the most comprehensive. The STOP-BANG questionnaire is a questionnaire frequently used by anesthesiologists to investigate OSAS in preoperative evaluation.[8] The Berlin sleep questionnaire is a questionnaire designed for community screening of OSAS.[24] In cases where OSAS persists despite CPAP treatment and if OSAS that disappears with CPAP treatment develops again, the adequacy of sleep duration should be ensured. There may be different conditions such as misdiagnosis of OSAS, incorrect CPAP titration, inadequate CPAP compliance, inadequate sleep hygiene, depression, narcolepsy, idiopathic hypersomnia, other sleep disorders, secondary acquisition. Other conditions associated with residual sleepiness despite CPAP therapy include depression, narcolepsy with cataplexy, narcolepsy without cataplexy, idiopathic hypersomnia, behaviorally induced insufficient sleep syndrome, Kleine-Levin syndrome, hypersomnia associated with menstruation, hypersomnia due to drug or substance use, circadian rhythm sleep disorder (shift work disorder, delayed sleep phase disorder, early sleep phase disorder, irregular sleep-wake rhythm.[12]

POLYSOMNOGRAPHY

PSG is the gold standard in the diagnosis of OSAS. PSG is the process of simultaneous and continuous recording of respiratory, cardiovascular, neurophysiologic and other physiologic parameters during sleep, usually throughout the night. With these channels, the presence of apnea, apnea duration, apnea type (obstructive/central) and the stage of sleep are evaluated. It is a very expensive, time-consuming study that requires trained personnel and specialized equipment.[3,6,12] When analyzing the PSG recording, the patient's sleep, respiratory and arousal scoring, leg movements during sleep and other pathologies are scored. In addition to normal parameters, snoring sounds, intrapleural pressure, pulmonary artery pressure, and arterial blood gas values can be measured optionally.[3,4,25] Anterior tibial muscle EMG is important in determining restless leg syndrome by examining periodic leg movements.[7,25] As a result of many developments in recent years, automatic diagnosis of OSAS patients is made using polysomnography data, signal processing and artificial intelligence methods.[3,9]

As a result of polysomnographic study, the value obtained by dividing the sum of the number of apneas and hypopneas in sleep by the duration of sleep in hours is called the apnea hypopnea index AHI. The grading of OSAS is based on the AHI value determined as a result of PSG.[4,7] The AHI value is measured by the number of apnea episodes that occur due to oxygen desaturation in a certain period of sleep. According to this index, AHI<5 is considered as non-OSAS, 5< AHI< mild, 15< AHI <30 moderate, AHI >30 severe OSAS (Table 2).[6-8]

Table 2. Characteristic PSG* findings for OSAS

- Frequent recurrent apneas, hypoppneas and waking reactions
- Increase in superficial sleep duration, decrease in deep sleep and REM** sleep duration
- Frequent recurrent episodes of oxygen desaturation
- Increase in frequency and duration of apneas and degree/duration of oxygen desaturation
- Typically, paradoxical chest and abdominal movements during apnea
- Bradycardia during apnea, tachycardia and arrhythmias afterwards
- Loud and irregular snoring interrupted by frequent recurrent episodes of apnea

*PSG: Polysomnography, **REM: Rapid eye movement, OSAS: Obstructive sleep apnea syndrome

AUXILIARY DIAGNOSTIC METHODS

Although PSG is the gold standard method for the diagnosis of OSAS, auxiliary diagnostic methods are utilized because they are time consuming and expensive.[9,12] These methods include chest radiography, thoracic tomography, magnetic resonance, nasopharyngolaryngoscopy, blood and urine tests, pulmonary function tests and arterial blood gas examinations.[3,4]

TREATMENT

There are various alternatives in treatment. These include weight loss, avoidance of alcohol and sedatives, exercise, changing the lying position, intraoral device (IOD), nasal

positive airway pressure (CPAP-Continuous positive airway pressure) and surgical treatment (Table 3).

Table 3. OSAS treatment

General precautions	
• Treatment for risk factors • Treatment of concomitant diseases* • Warning about traffic and work accidents • Weight loss	• Medical • IOD** • CPAP *** • Surgical • Combination

OSAS: Obstructive sleep apnea syndrome, *Hypotroism, acromegaly,diabetes mellitus, excessive androgen release, respiratory diseases, neurological and cardiovascular diseases, **IOD: Intraoral device, ***CPAP: Continuous positive airway pressure

The gold standard treatment method in OSAS is CPAP application. The aim of CPAP treatment is to achieve an AHI <5 and to eliminate the most common symptoms (snoring, apnea with apnea EDS). It is mainly applied to patients with moderate and severe OSAS with AHI >15. However, CPAP treatment is recommended in patients with mild OSAS with AHI between 5 and 15 who have significant symptoms and/or cardiovascular and cerebrovascular risk factors. The mechanism of action of CPAP, which has been the most important treatment of OSAS for the last 20 years, is the view that it prevents URS collapse like a kind of stent and prevents apneas by maintaining patency.[12,26] This theory was first proposed in 1981 by Sullivan et al.[4] Although some studies have shown that CPAP leads to an increase in lung volume, which increases the stabilizing effect of the upper airway, it has been shown that inflammation and cytokines in OSAS can be reduced by CPAP treatment.[12] In CPAP treatment, EDS may persist despite an AHI <5. The persistence of EDS despite optimal CPAP treatment is called residual sleepiness.[12] Modafinil and armodafinil are effective in the treatment of residual sleepiness in OSAS patients using CPAP. Long-term CPAP therapy significantly reduces diurnal sympathetic tone in OSAS patients. With CPAP treatment, 5% OSAS patients may experience persistent sleepiness.[26] With weight loss, patients with OSAS experience a decrease in AHI and improvement in sleep quality. Prevention of supine sleeping has been shown to improve sleep-disordered breathing in mild position-dependent OSAS patients. There is no specific pharmacologic agent used. Although partial responses have been obtained to the most studied drugs such as proptilin, medroxyprogesterone and acetazolamide, the current accepted opinion is that drugs are ineffective in the treatment of OSAS. OID treatment is generally applied in patients with simple snoring or mild OSAS, in moderate and severe OSAS in whom CPAP cannot be applied, in patients who are candidates for tonsilloctomy, adenoidectomy, tracheostomy but refuse this attempt, in upper URS resistance syndrome, after failed uvulopalatopharyngoplasty (UPPP) operation. Polysomnography should be performed before starting and during the follow-up of OID treatment. OID are known as devices that keep the tongue in front and advance the mandible forward.[11,26]

The surgical method of treatment is UPPP, which includes the elimination of nasal problems, soft palate and tonsils. The success rate of UPPP operation is around 90% for snoring and 50% for apnea. For simple snoring and mild apnea, a method called somnoplasty, which works with radiofrequency waves, is also used.[9]

Regarding driving safety, it has been stated that people without excessive sleepiness and with an AHI of less than 20 per hour are eligible for a commercial license without treatment for OSAS. It has been found that untreated OSAS patients cause 2-7 times more traffic accidents than the normal population. According to the revised Turkish legislation dated 29.12.2015, applicants with a BMI >33 kg/m^2 at the time of driver's license renewal or new application must undergo a full night PSG test regardless of their symptoms. Rigorous assessment of driver's license applicants with a BMI above 33 kg/m^2 can play an important role in improving safety on the road.[27]

COMPLICATIONS

OSAS triggers neurophysiologic reactivations including an increase in systemic inflammatory response, plasma adrenocorticotropic hormone, plasma norepinephrine and a decrease in growth hormone concentrations (Table 4).[28]

Table 4. Complications in OSAS

Cardiovascular system	Systemic hypertension Cardiac arrhythmias Left heart failure Ischemic heart disease Pulmonary hypertension Right heart failure
Pulmonary system	Overlap syndrome Bronchial reactivity
Neurological system	Cerebrovascular disease Excessive daytime sleepiness Morning headache Nocturnal epilepsy Restlessness
Cognitive impairment	Depression Anxiete
Endocrine system	Decreased libido Impotence
Nephrological	Nokturi Nocturnal enuresis
Gastrointestinal system	Gastroesophageal reflux
Hematologic system	Secondary polycythemia
Socioeconomic	Traffic and work accidents Economic losses Decreased quality of life Glaucoma Hearing loss Sudden death

OSAS: Obstructive sleep apnea syndrome

With the increase in negative intrathoracic pressure during OSAS, right ventricular return increases, the right ventricle is distended, the interventricular septum is displaced to the left and left ventricular filling is prevented, thus decreasing the stroke volume. In addition, left ventricular afterload increases with overstimulation of the sympathetic nervous system and cardiac output decreases. The risk of congestive heart failure more than doubles in OSAS. Recurrent hypoxemia during sleep, systemic hypertension and increased sympathetic activity facilitate the development of atherosclerosis.[19] Systemic hypertension is seen in 30-50% of OSAS patients. Pulmonary hypertension may develop as a result of hypoxic pulmonary vasoconstriction and remodeling. The incidence is 20-41.[8]

It has been found that there is a progressive increase in systemic blood pressure during the day and during sleep in patients with OSAS.[14] Sleep apnea and hypopnea episodes cause transient blood pressure changes including an increase of 30 mmHg or more in arterial blood pressure. Changes in renin-angiotensin and vasoactive paptides and insulin resistance syndrome, especially in overweight sleep apnea patients, may also increase the occurrence of hypertension.[29] Patients with a history of coronary angioplasty, coronary artery bypass, cerebrovascular

events or acute coronary syndrome have been found to have a positive association with OSAS.[30]

OSAS is more common in COPD patients with high BMI.[16,31] The association of respiratory system diseases (COPD, asthma, cystic fibrosis and interstitial lung disease) with OSAS is known as overlap syndrome. CPAP therapy controls nocturnal attacks in asthma and reduces bronchial hyperresponsiveness.[32] The presence of inflammatory cytokines in the peripheral circulation of patients with OSAS and the effects of superoxide radicals activate immune and inflammatory pathways, leading to asthma and COPD.[8,21]

Tissue hypoxia and associated oxidative stress and inflammation products that occur during OSAS lead to endothelial damage and metabolic irregularities.[33] Studies have found a relationship between OSAS and snoring and DM (diabetes mellitus). Sleep quality is impaired in most patients with type 2 DM. This is usually related to DM type 2 and cardiovascular diseases.[34]

As a result of tissue hypoxia, cerebral blood flow changes and stress mechanisms are stimulated. Neurohumoral and autonomic activation leads to disturbances in glucose metabolism and the release of proinflammatory cytokines such as TNF-alpha, IL-1 and IL-6. Insulin resistance is around 20% in OSAS. The metabolic disorder is expected to improve 3 months after the start of CPAP use.

Sleep interruptions have been shown to increase blood cortisol levels. Growth hormone, which is normally released in slow wave sleep, is suppressed in OSAS patients due to a decrease in deep sleep. Due to less and inefficient sleep, leptin level decreases, ghrelin level and appetite increase and obesity increases. A strong association has been found between OSAS and some psychiatric disorders such as post-traumatic stress disorder, anxiety, anxyiete and depression. OSAS impairs cognitive stages such as memory, thinking and perception.[15,35] Accelerated tumor angiogenesis and high oxidative stress observed in nocturnal hypoxia have been reported to promote possible tumor formation by damaging DNA and RNA, and severe OSAS has been reported to cause an increase in cancer-related mortality.[8]

Decreased REM sleep duration was found to be statistically significant in patients with severe OSAS.[7] It was found that the percentage of sleep efficiency and the percentage of REM sleep decreased and sleep duration increased in these patients. Periodic leg movements (PBMs) are involuntary, repetitive, sterotypic, short-term, segmental and often affecting the lower extremities during sleep. High AHI values are associated with PBD.[7,12] Lying in supine position during rapid eye movement (REM) and non-REM sleep causes sleep-disordered breathing in most cases. In OSAS, changing from a ninety-degree sitting to a lying position narrows the pharyngeal opening in both apneic and normal individuals due to the effect of gravity. A decrease of at least 50% in AHI occurs in the transition from supine to lateral position.[25] The currently accepted view is that patients should be advised to lie in the lateral position and no additional measures should be taken. Placing a tennis ball, backpack or sandbag on the patient's back so that the patient is uncomfortable when turning over is no longer on the agenda.[11]

Since erythropoietin does not decrease during sleep in OSAS patients, secondary polycythemia is seen in 10%.[36]

In OSAS patients, atrial natriuretic paptide (ANP) release increases due to stretching of the right atrial wall caused by fluctuations in intrapleural negative pressure. ANP increases urinary and sodium excretion by suppressing the renin-angiotensin-aldeserone system. Nocturia is very common in this group with a rate of 28%.[37]

Increased respiratory effort and increased gastric pressure with abdominal pressure cause gastroesophageal reflux. Intermittent hypoxemia may lead to hyperlipidemia and hepetosteatosis.[38]

Many neuropsychiatric complications such as depression, anxiety, cognitive impairments, memory impairment, forgetfulness, personality changes, amnesia, and difficulty in concentration may be observed. The incidence of morning headache, nocturnal epilepsy and cerebrovascular disease may increase.[35]

OSAS is one of the causes of sudden death during sleep. Malignant arrhythmias, ischemic heart diseases, heart rate changes and acute myocardial infarction may cause sudden death.[39]

PROGNOSIS

The risk of morbidity and mortality increases when OSAS is severe. It is generally thought that this is a consequence of asphyxia and wakefulness reactions during apnea-hypopnea formation. Patients are lost due to cardiac arrhythmias, transient pulmonary artery pressure changes and post-apnea hypoxemia, especially during the REM period of sleep. Sudden deaths may also occur due to cardiac and cerebral causes related to hypertension. Traffic and occupational accidents in OSAS patients are another reason that increases mortality.[7,27]

CONCLUSION

OSAS is a common but understudied disorder in the population. It is characterized by recurrent complete or partial upper airway obstruction during sleep, leading to recurrent oxygen desaturations, cyclic adverse changes in heart rate, blood pressure and sympathetic activity, and disruption of sleep structure. Dentists evaluating the anamnesis and clinical examination findings can serve as the first line of care. Considering that this disease is at the root of serious cardiovascular and cerebrovascular diseases such as hypertension and cardiac arrhythmias, early diagnosis and treatment are crucial in improving the quality of life of patients and reducing morbidity and mortality. Supportive studies are needed to find new biomarkers, genetic risk assessment, and to determine the specific pathology of the patient.

ETHICAL DECLARATIONS

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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